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Case Report

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Severe Ataxia Secondary to Copper Deficiency Myelopathy in a Patient with History of Gastric Bypass Surgery: A Case Report

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Abstract

This is a 46-year-old female with significant past history notable for systemic lupus erythematous, gastric bypass surgery, and anterior cervical diskectomy and fusion at cervical 5-6 who presents to clinic with months of progressive difficulty with ambulation and falls. Patient initially misdiagnosed with severe cervical stenosis and then vitamin B12 deficiency as the cause of her gait ataxia. Subsequent workup later proved patient was suffering from copper deficiency leading to subacute combined degeneration of the spinal cord. Copper deficiency is a rare and often overlooked cause of spinal cord injury especially in the context of gastric bypass and zinc supplementation for wellness in today's COVID era. More attention should be placed on copper deficiency as a root cause of gait ataxia.

Keywords: Myelopathy; Cervical Stenosis; Copper Deficiency; Gastric Bypass; Case Report

What is known/new: Copper deficiency is underdiagnosed and a rare cause of myelopathy. With the increased prevalence of health supplements in the post-COVID era, oral zinc supplementation can be a potential agent in copper deficiency.

Case Study

A 46-year-old female presented to an outpatient spine clinic with chief complaints of brain fog, memory recall issues, gait ataxia, and increasing falls. Her medical history included Roux-en-Y gastric bypass, Lupus treated with Myfortic, generalized anxiety disorder, and a previous anterior cervical diskectomy and fusion (ACDF) at C5-6. Physical exam was notable for 4/5 motor strength in the upper and lower extremities, wide-based gait, inability to do tandem walking, finger-to-nose dysmetria, and positive Romberg's sign. Notably, the patient had a negative Hoffman's sign, and reflexes were hyporeflexive throughout.

Initial, the focus was possible adjacent segment disease, given the patient's history of ACDF. Magnetic resonance imaging (MRI) displayed moderate-to-severe central canal stenosis above the ACDF site at C4-5, suggesting compressive myelopathy. A closer examination of the MRI cervical spine T2 sequence displayed a hyperintense signal in the dorsal column that was not reported by the radiologist. The hyperintensity signal demonstrated an "inverted V-sign" on the T2 axial cut. (figure 1) Given her history of gastric bypass and recent discharge from the hospital, it was suspected to be subacute combined degeneration secondary to vitamin B12 deficiency.

Further metabolic panel analyst showed a B12 level of 497 pg/mL (range:

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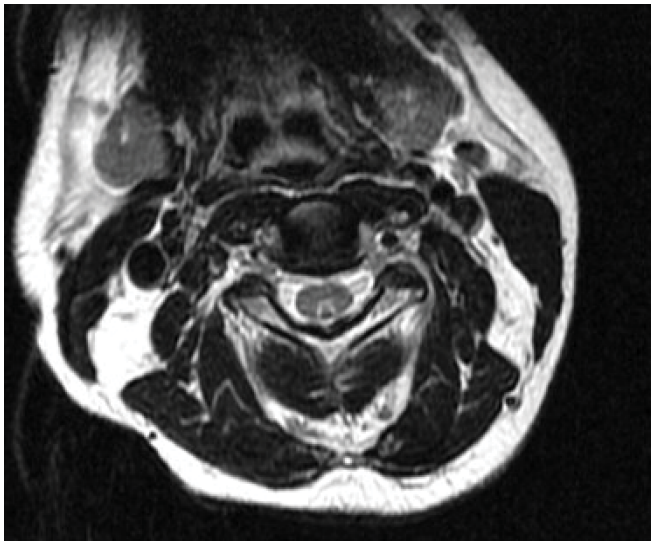

Figure 1: Inverted V-Sign on T2 Sequence Axial Cut of MRI Cervical Spine with Contrast.

213-816 pg/ML, but the patient has been as low as 274 pg/mL 2 months before that). Despite B12 levels in a low-normal range, her symptoms and MRI findings suggested a possible relative deficiency in vitamin B12. However, a normal methylmalonic acid (MMA) level contradicted the theory of a B12 deficiency (as MMA is elevated in B12 deficiency). This led to considerations of alternative diagnoses such as tabes dorsalis, HIV vacuolar myelopathy, diabetic myelopathy, and even copper (Cu) deficiency. Subsequent examination of her lab results unveiled low copper levels of 43 mcg/dL (range: 77-206 mcg/dL, although Cu level was <10 mcg/dL only 3 months prior to that). The patient was then admitted to the hospital for urgent copper repletion. During admission ceruloplasmin levels was checked and found to be low at 18.0 mgs/dL (range: 20-51) confirming copper deficiency. HIV and syphilis labs were non-reactive. The patient was supplemented with IV copper (cupric chloride) 2mgs/day x 7 days to normal levels. The patient has been in outpatient physical therapy since with marked improvement.

Copper deficiency is a rare and often overlooked consequence of gastric bypass surgery due to micronutrient malabsorption.1 Copper deficiency can also be caused by excessive oral zinc supplementation [1]. Serum zinc and copper concentrations are rarely measured in patients prescribed zinc supplementation, furthering delay in diagnosis of iatrogenic copper deficiency. Copper-level assessments should be considered when evaluating ataxia, as delay in diagnosis can lead to poor neurological outcomes. This deficiency led to severe demyelination of the dorsal column of the spinal cord, manifesting as ataxia and other neurological symptoms [2]. Case series have described it in patients with chronic gastrointestinal pathology [2]. During the COVID-19 pandemic, oral zinc supplementation was found to reduce the 30-day mortality ICU admission rate and shorten symptom duration, making PO zinc supplementation more commonplace in the post-COVID-19 era [3] Excessive zinc intake causes the upregulation of metallothionein zinc-binding protein in enterocytes, which have a high affinity for copper [4]. When the enterocytes are sloughed into the gastrointestinal tract, significant amounts of copper are lost and can eventually cause a copper-deficient state [4]. Copper is a trace metal that acts as a prosthetic group in several key enzymes and is thus essential for the structure and function of the bone marrow and nervous system [5].

Moreover, up to 10% of those undergoing Roux-en-Y gastric bypass surgery experience copper deficiency; however, sequelae are not frequently reported [6] There have been reports of non-compressive copper deficiency myelopathy mimicking subacute combined degeneration due to vitamin B12 deficiency [6] Copper deficiency myelopathy has mostly been described in the past ten years and represents a mimicker of subacute combined degeneration due to vitamin B12 deficiency [7]. Treatment typically leads to hematological normalization and neurological improvement or stabilization [7].

This case underscores the evolving landscape of medical care, particularly in the wake of the COVID-19 pandemic. With the emergence of evidence suggesting the benefits of oral zinc supplementation in managing COVID-19 symptoms, the risk of iatrogenic copper deficiency becomes increasingly pertinent. As illustrated in this case, excessive zinc intake led to the sequestration of copper in enterocytes, predisposing the patient to copper deficiency, manifested in her neurological presentation. Moving forward, healthcare providers should remain vigilant in assessing and managing nutrient deficiencies, especially in patients with a history of gastric bypass surgery or zinc supplementation. Failure to recognize and address copper deficiency can result in significant neurological sequelae, as evidenced by this patient's journey through multiple specialty clinics before receiving appropriate treatment.

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